that doubt the effectiveness of non-invasive techniques forget that it is their unfamiliarity that limits the value of myocardial perfusion imaging and not the technique itself. Instead of ignoring it they should follow the practice of their colleagues who are able to manage patients with objective measurements of myocardial perfusion rather than subjective impressions of the severity of disease made from the coronary arteriogram. They might then find that they can make many therapeutic decisions without the need to reach for their catheters, and we shall be spared the debate about the role of cardiac catheterisation in the district general hospital.

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1 Steinberg EP, Klag MJ, Bakal CW, Strauss HW, Boucher CA, Guiney TE. Exercise thallium scans: patterns of use and impact on management of patients with known or suspected coronary artery disease. *Am J Cardiol* 1987;**59**:50–5.
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Acromegalic heart disease

SIR,—We disagree with several of the statements in the article by Rodrigues *et al* entitled "Subclinical cardiac dysfunction in acromegaly: evidence for a specific disease of heart muscle" (1989;**62**:185–94).

In the summary Rodrigues *et al* say that "This is the first study to find evidence of subclinical cardiac diastolic dysfunction in acromegaly", and in the discussion they say that "Left ventricular diastolic function has not previously been studied in acromegaly". I would like to draw attention to the paper of Bertoni and Morandi entitled "Impaired left ventricular diastolic function in acromegaly: an echocardiographic study", where relaxation abnormalities of the left ventricle were shown in acromegalic patients in 1987.[1]

On the basis of the paper by Lie and Grossman,[2] Rodrigues *et al* suggest that the fibrosis which is partly responsible for the diastolic filling disturbance is a consequence of an inflammatory process. Van den Heuvel *et al* took biopsy specimens of the right ventricle of an acromegalic patient and later examined specimens taken at necropsy. Both specimens showed the same changes: hypertrophic myocardial fibres and some fibrous thickening of the endocardium. There was no evidence of any inflammatory changes.[3] In rats with myocardial hypertrophy caused by tumours producing growth hormone Gilbert *et al* found direct evidence that there was no underlying inflammatory process.[4]

These results prove that the main pathological background of acromegalic heart disease is left ventricular hypertrophy, with some contribution from myocardial fibrosis and the increased collagen content of the myocardium. We found the dilated form of the disease in a few of our patients but not an inflammatory process; these patients were burned-out cases with lower serum concentrations of growth hormone than those with hypertrophy.[5]

Rodrigues *et al* cited the second edition of Feigenbaum's *Echocardiography* (not published in 1979 but in 1976) as saying that echocardiography is an insensitive method of assessing left ventricular function. However, this is not the opinion given in the fourth edition published in 1986.[6] Radionuclide ventriculography is probably a better method of assessing the ejection fraction than echocardiography. But because there are several other indices of left ventricular function (for example, enlargement of the left ventricle and segmental wall movement abnormalities) echocardiography cannot be deemed to be an insensitive method of evaluating left ventricular function.[7]

In Rodrigues *et al*'s paper apart from 13 self-citations, there are only three references from 1982–84 and only one, an abstract, dated 1986. The references I cite were published between 1983 and 1987.

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1 Bertoni PD, Morandi G. Impaired left ventricular diastolic function in acromegaly: an echocardiographic study. *Acta Cardiol (Brux)* 1987;**15**:1–10.
2 Lie JT, Grossman SJ. Pathology of the heart in acromegaly: anatomic findings in 27 autopsied patients. *Am Heart J* 1980;**100**:41–52.
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This letter was shown to the authors, who reply as follows:

SIR,—In reply to Csanády's letter we would like to make the following comments. Bertoni and Morandi published a paper on left ventricular diastolic function in 1987,[1] but our study was largely completed in 1986 and was presented in part to the British Cardiac Society in December 1986.[2]

Csanády seems to disregard the seminal work on pathological findings of Lie and Grossman[3] in favour of the necrospy findings in a patient described by van den Heuvel *et al.*[4] Necropsy findings in only one patient do not disprove the findings of a large study. Furthermore, in our study we tried to assess the influence of myocardial hypertrophy by studying left ventricular mass derived echocardiographically and showed that there was no correlation between peak filling rate abnormalities and left ventricular wall thickness or left ventricular mass. Therefore, it is unlikely that the abnormal peak filling rate seen in our patients was merely a reflection of hypertrophy.

Though we agree that echocardiography can be used not only to measure ejection fraction but also to assess the anatomical consequences of left ventricular dysfunction, such as left ventricular enlargement and wall motion abnormalities, the point we made in our discussion was that subtle changes in diastolic relaxation were shown better by radionuclide ventriculography. Indeed, Feigenbaum[5] pointed out the drawbacks and pitfalls of calculating ejection fraction by cross sectional echocardiography and did not go into any detail about the assessment of subtle diastolic function by this technique. We referred to our previous validation studies in the text to establish the quantitative success and reproducible nature of the radionuclide technique for assessing subtle diastolic left ventricular dysfunction. Many methods have been used to study a large group of patients with a rare condition. Previous studies did not deal with the same questions, nor was thallium-201 imaging used to exclude obstructive coronary artery disease.[6] Thus our suggestion that radionuclide ventriculography is better than echocardiography for picking up subtle left ventricular diastolic dysfunction is valid.

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Severe haemolytic anaemia after replacement of the mitral valve by a St Jude medical prosthesis

SIR,—The interesting case report by Feld and Roth (1989;**62**:475–6) of severe haemolytic anaemia after mitral valve replacement with a St Jude medical prosthesis highlights the difficulty in detecting mitral paraprosthetic leaks. Despite clinical evidence of mitral regurgitation in their patient, careful echocardiography with Doppler studies failed to detect any prosthetic abnormality and left heart catheterisation was required to show severe paraprosthetic regurgitation.

We found that transoesophageal echocardiography with a 5 MHz phased array transoesophageal transducer (HP 21362A)[1] was helpful in nine patients (mean age 58 years) with mitral prosthetic regurgitation. One of them also had severe haemolytic anaemia after the insertion of a 27 mm St Jude mitral prosthesis. Mitral regurgitation was detected by transthoracic echocardiography, including Doppler studies and colour flow mapping, in only six patients. Transoesophageal echocardiography clearly showed paraprosthetic leaks in all six patients with a mechanical prosthesis and central leaks due to leaflet dehiscence in all three patients with a bioprosthesis. These results were confirmed by cardiac catheterisation or operation or both in five patients, with two patients proceeding to surgery on the basis of transoesophageal echocardiographic findings alone.

The evaluation of prosthetic mitral valve dysfunction by transthoracic echocardiography is limited by acoustic masking by the prosthesis, whereas the transoesophageal probe can be positioned directly behind the left atrium. The results of transoesophageal echocardiography of mitral prosthetic dysfunction correlate well with surgical findings.[2 3]

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1 Seward JB, Khandheria BK, Oh JK, *et al.* Transesophageal echocardiography: technique, anatomic correlations, implementation and clinical applications. *Mayo Clin Proc* 1988;**63**:649–80.
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Follow up of patients treated with balloon dilatation of the aortic valve

SIR,—Vogel *et al* (1989;**62**:148–53) used pressure differences measured invasively as peak to peak gradients and non-invasively by Doppler echocardiography as peak instantaneous gradients to evaluate the success of treating aortic valve stenosis by balloon dilatation. They used a regression equation obtained by comparing the results in 12 patients to correct Doppler gradients to the corresponding peak to peak gradients. There are several objections to this approach:

(a) Peak to peak gradients measured by catheterisation and peak instantaneous gradient measured by Doppler echocardiography are conceptually different and the use of regression equations to estimate one from the other is not reliable.[1] Large differences are frequently seen between these two types of gradient.[2 3]

(b) This difference is often considerable in patients with aortic regurgitation,[2] which was present in 60% of the patients in this study after the procedure.

(c) The peak instantaneous gradient is always higher than the peak to peak gradient.[3] The regression equation applied showed that the inverse was the case in the reported study, indicating less than perfect Doppler echocardiographic recordings.

If pressure gradients are used it is probably more appropriate to estimate either mean gradients or peak instaneous gradients, both of which can be measured invasively as well as by Doppler echocardiography. This eliminates the need for a regression equation.

The use of valve areas has been recommended for follow up of adult patients.[4] This is probably more important in children, in whom the absolute valve areas vary more than in adult patients. I recommend use of the continuity equation to calculate the valve areas according to Skjaerpe *et al*[5] using the actual transvalvar flow.

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1 Currie PJ, Seward JB, Reeder GS, *et al.* Continuous-wave Doppler echocardiographic assessment of severity of calcific aortic stenosis: a simultaneous Doppler-catheter correlative study in 100 adult patients. *Circulation* 1985;**71**:1162–9.
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This letter was shown to Dr Benson, who replies as follows:

SIR,—Dr Dag Teien's comments prompted us to review our study and we agree with him that peak to peak gradients are poor reflections of instantaneous gradients across the stenosed aortic valve.[1] The instantaneous gradient generally does overestimate the peak to peak gradient. Our regression equation was obtained at the same time as direct measurements of aortic and left ventricular pressures in the catheterisation laboratory. Upon reflection, it has become clear to us that under these conditions alterations in pressure and flow dynamics, systolic ejection times, and arterial compliance can influence the contour of the upstroke of the aortic pressure curve in such a way as to have the peak to peak gradient approach the instantaneous gradient. We no longer use the regression equation to correct the pressure gradient estimate.

Because we did not find depressed pump function in children with aortic stenosis we still regard the left ventricular to aortic gradient as clinically useful in decision making. This view is supported by the studies of the course of untreated aortic stenosis[2] in the paediatric patients in which peak to peak gradients rather than valve areas were used for assessment. Furthermore, from infancy to adolescence valve areas change considerably, in a non-linear way, which additionally complicates estimating a normal valve area for a given patient.

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1 Gordon JB, Folland ED. Analysis of aortic valve gradients by transseptal technique, implications for noninvasive evaluation. *Cathet Cardiovasc Diagn* 1989;**17**:144–51.
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BOOK REVIEWS

Electrocardiography of Arrhythmias. Charles Fisch. (pp 451; £34.03.) Philadelphia and London: Lea and Febiger, 1990. ISBN 0-8121-1265-2.

Does the surfeit of books on basic electrocardiography—introductions, primers, guides and the like—tell us that the subject is unduly difficult to comprehend? Perhaps it is too much taken for granted or possibly ignored by teachers; certainly in the United Kingdom, the home of Waller and Lewis two of the founding fathers, this seems to be so. We have no tradition of the "heart station" with reporting of tracings as a regular feature of hospital life and thus less opportunity for the watching student to learn the practical side of reporting from a mentor.

The European masters did know their fundamentals, however, though many had to make their way to the United States in the years encompassing the second world war. A happy liaison then developed between the rising expert on arrhythmias at the Michael Reese Hospital in Chicago, Louis N Katz, and two immigrants from Prague, Richard Langendorf and Alfred Pick. From them came a succession of papers and books on arrhythmias that have become classics. And now we have a major contribution from one of their foremost disciples and colleagues, Charles Fisch of Indianapolis. Dr Fisch is particularly well known for the courses he conducts at his Krannert Institute; but his programme on advanced arrhythmias on the Sunday before the annual meeting of the American College of Cardiology has brought him to the attention of many more who crowd into the lecture theatre to learn from his incisive analyses carried out in the form of a dialogue with members of a team to whom he shows complex tracings.

Now he has assembled and analysed representative selections from his extensive collection of material in the form of this book. It is not for the beginner, who might be better advised to have at hand and consult something like Dunn and Lipman's *Lipman-Massie Clinical Electrocardiography* (which also reflects the approach of the Chicago school) as well as a recommended introductory text. What Fisch does provide is an essential code book that explains concepts and phenomena which must be understood if the electrocardiographic analysis of arrhythmias—simple as well as complex—is to be more than the memorisation of some patterns.

Here we find descriptions of events that influence the electrocardiogram in classic, important, but too often poorly comprehended ways. The chapter headings indicate that this is no ordinary book that starts at the sinus node and ends with the recovery of the ventricles. They show the reader what is so often missing from the more conventional texts: careful descriptions of, for example, concealed conduction, aberration, and entrance and exit block that make possible understanding of what otherwise seems so difficult to sort out.

Although the text is liberally explained by well annotated examples of conventional elec-